CLINICAL STUDY ON CELLULITIS

DISSERTATION SUBMITTED TO

In partial fulfillment of the requirement for the degree of

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DEPARTMENT OF GENERAL SURGERY

TIRUNELVELI MEDICAL COLLEGE

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Dear , Dr. Pradeep Rao, The Tirunelveli Medical College Institutional Ethics Committee (TIREC) reviewed and discussed your application during the IEC meeting held on 05.08.2016.

THE FOLLOWING DOCUMENTS WERE REVIEWED AND APPROVED

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2. Study Protocol ☐
3. Department Research Committee Approval ☐
4. Patient Information Document and Consent Form in English and Vernacular Language ☐
5. Investigator's Brochure ☐
6. Proposed Methods for Patient Accrual Proposed ☐
7. Curriculum Vitae of the Principal Investigator ☐
8. Insurance /Compensation Policy ☐
9. Investigator's Agreement with Sponsor ☐
10. Investigator's Undertaking ☐
11. DCGI/DGFT approval ☐
12. Clinical Trial Agreement (CTA) ☐
13. Memorandum of Understanding (MOU)/Material Transfer Agreement (MTA) ☐
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This is certify that this dissertation work title "**CLINICAL STUDY ON CELLULITIS"** of the candidate **DR.R.PRADEEP RAO,** with registration Number **221611357** for the award of **M.S.** Degree in the branch of **GENERAL SURGERY (I).** I personally verified the urkund.com website for the purpose of plagiarism check. I found that the uploaded thesis file contains from introduction to conclusion page and result shows **2 percentage** of plagiarism in the dissertation.

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https://en.wikipedia.org/wiki/Cellulitis
https://vdocuments.site/documents/cellulitis-guidelines-crest-05.html
https://vdocuments.mx/documents/cellulitis-guide.html

Instances where selected sources appear:

8

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INTRODUCTION

Cellulitis is a non-suppurative, invasive infection caused by bacteria that is characterized by specifically affecting the dermis and subcutaneous fatty layer by a normal skin flora or an exogenous bacteria.

Cellulitis in Latin: cellula means– cell,itismeans –inflammation and its subtype erysipelas is a greek word, erythrós means- red,pella means- skin, are among the most common condition requiring hopitilization**.**

In contrast to cellulitis, erysipelas is a streptococcal bacterial infection of superficial layers of the skinis characterised by sharp demarcation, a palpable edge and salmon-red erythema and is accompanied by high fever.

Although cellulitis can affect any part of the body the most common sites involved are upperlimb, lowerlimb and face.

The lowerlimbs are affected commonly following a breach due to cracks, breaks, blisters, surgical wounds, ulcers in the skin.These breaks, need not be visible. Group A Streptococcus and Staphylococcus are the normal flora of the skin, but normally will not cause any infection until the skin surface is intact.

Other risk factors include diabetes,obesity, old age, immune compromised individuals, insect bite, animal bite, tattoos, injecting drugs (especially subcutaneous or intramuscular injection), pregnancy.

Diabetics are the most common risk factors for the leg cellulitis mainly because of the poor blood sugar control ,bare foot walking resulting in trauma and immunocompromised. The growth of the organism in the ulcers will cause cellulitis because of poor glycemic control status.

Non diabetics, renal failure, congestive cardiac failure are also prone for the development of cellulitis and its complications.Grade IV serious infections necrotizing fasciitisor underlying bone infectionshould be ruled out**.**

Grade 1 cellulitis can be treated as out patient department with oral

Analgesics, oral antibiotics and treating the cause.

But cellulitis ofgradesII, grade III, grade IV, presenting with systemic complication and various other comorbidities requires hospital stay, IV antibiotics and any surgical management.

AIM OF THE STUDY

1. To study the age and sex distribution, risk factors, clinical presentation, pathology of cellulitis admitted in Government Tirunelveli medical college Hospital from August 2017 to February 2018.
2. To study the causative agents responsible for cellulitis and theirsensitivity pattern.
3. To study the grading of cellulitis.
4. To study arterial and venous pathology and osteomyelitic changes in bones of cellulitis.
5. To study the outcome and benefit of the different treatment modalities of cellulitis.
6. To brief the resultant wound management after treating the condition.

REVIEWOF LITERATURE

Cellulitis is a non-suppurative, invasive infection caused by bacteria that is characterized by specifically affecting the dermis and subcutaneous fatty layerwithout the abscess or purulent discharge, or osteomyelitis changes orosteolytic changes and fasciitis.

Now a days cellulitis is defined as suppurartive lesion with complications like frank abscess formation, ulcerations, myositis, fasciitis and osteomyelitic changes.

Cellulitis is a spreading infection that's spread under the skin ,subcutaneous fat and underlying soft tissues. It can infect through damaged or broken skin. Cellulitis in lowerlimb with signs and symptoms warmth, pain, and swelling are the usual presentations[1,6].

This reddened skin or rash may signal a deeper, more serious infection of the inner layers of skin. Once below the skin, the bacteria can spread rapidly, entering the lymph nodes and the bloodstream and spreading throughout the body. This can result in influenza-like symptoms with a high temperature and fever, sweating and hypotension. Necrotizing fasciitis, also known by "flesh-eating bacteria", is a deep-layer infection which needs emergency surgical interventions.

Orbital cellulitis a subtype of facial cellulitis common in infants. Lower limbs are the most commonly involved sitesin adults as the leg is typically affected following a breach due to cracks, breaks, blisters, surgical wounds, ulcers in the skin which is the site of the portal of infection[2].

Diabetic population prone to cellulitis than the general population because of impaired immune system and blood circulation in the legs, poor glycemic controls which allows bacteria to grow more rapidly in the affected tissue, and end up in septicaemia if the infection enters the bloodstream. In diabetic neuropathy the ulcers are not painful and get infected.

Cellulitis, ulcers and soft tissue infections not uncommon in the non-diabetics, and they are in rapidly in increasing trend.

LOWER LIMB ANATOMY

Lower limb involves the pelvic bone, hip bone, buttocks, thigh, lower leg and the foot. It is mainly locomotor organ. The major bones of the leg are the long bones- femur (thigh bone), tibia (shin bone), and adjacent fibula. The patella is the sesamoid bone in front of the knee. Most of the leg skeleton has bony prominences and margins that can be palpated and are the anatomical landmarks[3]. These landmarks are the anterior superior iliac spine, the greater trochanter, the superior margin of the medial condyle of tibia, and the medial malleolus.

VASCULAR ANATOMY

The arteries of the lower limb are divided into a series of segments.

In the pelvis, the descending aorta, divides into a pair of common iliac artery. The common iliac artery splits into the internal and external iliac arteries.The external iliac artery descends along the medial border of the psoas major to exits the pelvis area under the inguinal ligament[1].

In the thigh, the external iliac artery enters the thigh as the femoral artery that descends the medial side of the thigh to the adductor canal.

The artery leaves through the adductor hiatus and becomes the popliteal artery.

Obturator artery, branch from Internal iliac artery also supplies the thigh.

The gluteal and thigh regions are supplied mainly by superior gluteal artery, internal pudendal artery, inferior gluteal artery and the perforating arteries of the thigh.

On the back of the knee the popliteal artery runs through the popliteal fossato the popliteal muscle where it divides into anterior and posterior tibial arteries.

In the leg, the anterior tibial artery enters the extensor compartment nearthe upper border of the interosseous membrane to descend between the tibialis anterior and the extensor hallucis longus and enters the foot through extensor retinacula of the foot and it becomes the dorsalis pedis artery of the foot.

The posterior tibial artery formed from the popliteal artery as continuation and gives common peroneal or the fibular artery.

THE VENOUS SYSTEMS ARE SUBDIVIDED INTO THREE SYSTEMS.

The deep veins, superficial veins, and perforator veins.

The venous valves assist in unidirectional blood flow from superficial veins to deep veins.

SUPERFICIAL VEINS:

- Greater saphenous vein
- Small saphenous vein

DEEP VEINS:

- Femoral vein
- Popliteal vein
- Anterior tibial vein
- Posterior tibial vein
- Fibular vein

MUSCULAR COMPARTMENTS OF THE LOWER LIMB

The fascial compartments of thigh divided into three - medial, lateral and posterior compartments, each has a group of muscles and nerve supplying the group.

The medial compartment are Adductors muscles - adductor longus, adductor magnus, adductor brevis, gracilis. The nerve innervating these muscle is the obturator nerve.

The posterior compartment are Flexors of the knee and extensors of the hip- Biceps femoris, semitendinosus, semimembranosus. The nerve innervating these muscles are sciatic nerve, more specifically the tibial nerve.

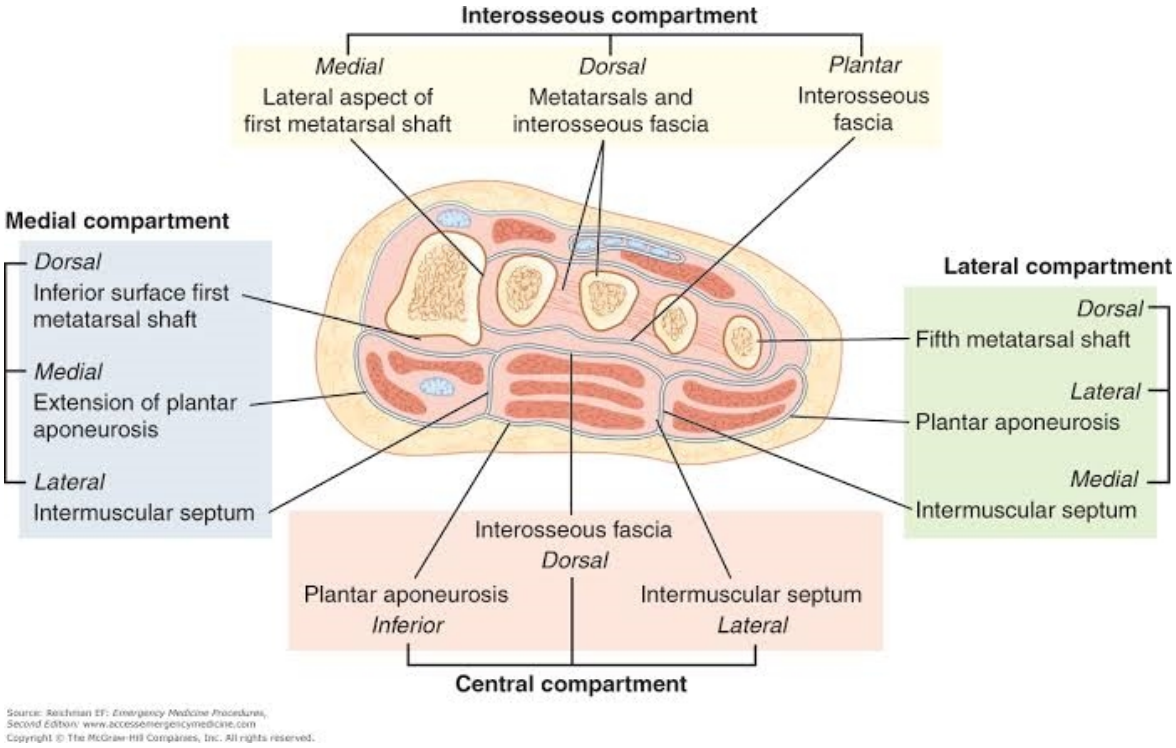
The anterior compartment are flexors of the hip and Extensors of the knee - sartorius and quadriceps. The nerve innervating these muscles are femoral nerve.

The leg has four fascial compartments.

COMPARTMENTS	MUSCLES	NERVES	BLOOD SUPPLY
Anterior	Tibialis anterior, Extensor hallucis longus, Extensor digitorum longus, Peroneus tertius.	Deep peroneal nerve	Anterior tibial vessels.
Lateral	Fibularis/peroneuslongus, andFibularis/peroneus brevis.	Superficial peroneal nerve.	
Deep posterior	Tibialisposterior, Flexor hallucis longus, Flexor digitorum longus, Popliteus.	Tibial nerve	Posterior tibial artery
Superficial posterior	Gastrocnemius, Soleus, Plantaris	Medial sural cutaneous nerve.	

The foot is composed of five compartments.

COMPARTMENT	BOUNDARIES
The interosseous compartment	Medially- Lateral first metatarsal. Dorsally- Metatarsal bones and the dorsal interosseous fascia. In plantar aspect- Plantar interosseous fascia
The lateral compartment	Medially- Intermuscular septum Laterally- plantar aponeurosis. Dorsally - Shaft of the fifth metatarsal bone,
The central compartment	Laterally and medially- Intramuscular septum Dorsally - Interosseous fascia, In plantar aspect- plantar aponeurosis.
Medial compartment	Medial- Plantar aponeurosis extension \ Lateral- Intramuscular septum Dorsally – Inferior surface of metatarsals
Calcaneal compartment	Quadrates plantae muscles.



RELEVANT ANATOMY OF UPPERLIMB

The upper limb is the region in a human extending from the deltoid region up to and including the hand, including the arm, axilla and shoulder.

Superficial muscles of the armTriceps is the major extensor and brachialis and biceps the major flexors. Biceps is, however, the major supinator and while performing this action it ceases to be an effective flexor at the elbow.

Muscles of posterior compartment of arm

- Triceps brachii, anconeus

Muscle of anterior compartment of arm

- Brachialis, biceps brachii

Muscle of forearm

Posterior

- Superficial - extensor digitorum, extensor digitiminimi, extensor carpi ulnaris.
- Deep- supinator, abductor pollicis longus,

extensor pollicis brevis, extensor pollicis longus, extensor indicis.

Anterior

- Superficial- pronator teres, flexor digitorumsuperficialis, flexor carpi radialis, flexor carpi ulnaris, palmaris longus.
- Deep- flexor digitorumprofundus, flexor pollicis longus, pronator quadratus.

Radial muscles

- Brachioradialis, extensor carpi radialis longus, extensor carpi radialis brevis.

MUSCLES OF THE HAND

Metacarpalmuscle

- Lumbricals, palmar introssei, dorsal interossei

Thenar muscle

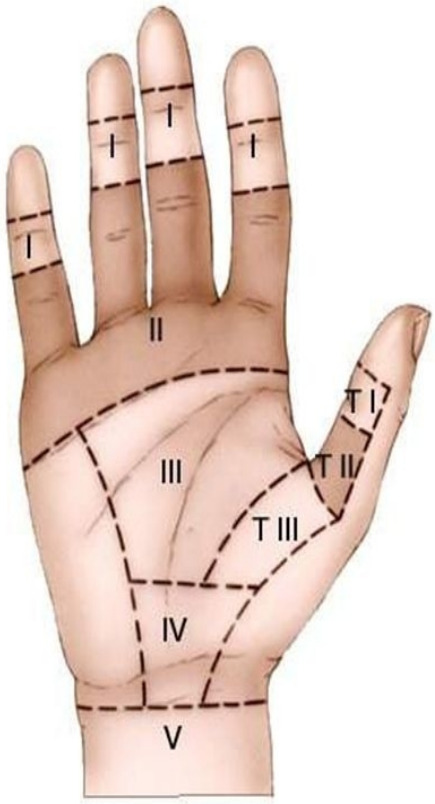
- Abductor pollicis brevis, adductor pollicis, flexor pollicis brevis, opponenspollicis

Hypothenar muscle

- Abductor digitiminimi, flexor

  digitiminimi, opponensdigitiminimi, palmaris brevis

ZONES OF HANDS



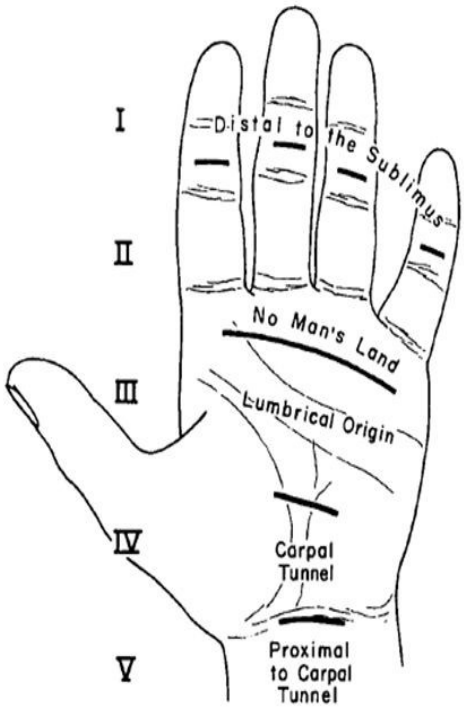


FIG. 1. So-called zones of the flexor tendons. Zone I: Distal to superficialis tendon; Zone II: Distal palmar crease to PIP crease; Zone III: Palm; Zone IV: Within the carpal tunnel; Zone V: Forearm proximal to the carpal tunnel. (Kleinert, H. E., Stormo, A.: Primary repair of flexor tendons. Orthop. Clin. North Am., 1973.)

Zone I: Distal to superficialis tendon

Zone II: Distal palmar crease to Proximal Inter Phalanges crease

Zone III: Palm

Zone IV: Within the carpal tunnel

Zone V: Forearm, proximal to the carpal tunnel.

- Motor innervation of upper limb by the five terminal nerves of the brachial plexus.
- The musculocutaneous nerve innervates all the muscles of the anterior compartment of the arm.
- The median nerve innervates all the muscles of the anterior compartment of the forearm except flexor carpi ulnaris and the ulnar part of the flexor digitorum profundus. It also innervates the three thenar muscles and the first and second lumbricals.
- The ulnar nerve innervates the muscles of the forearm and hand not innervated by the median nerve.

The axillary nerve innervates the deltoid and teres minor.

The radial nerve innervates the posterior muscles of the arm and forearm.

Arteries of the upper limb:

The superiorthoracic, thoracoacromial, posterior circumflex humeral and subscapular branches of the axillary artery.

The deep brachial, superior ulnar collateral, inferior ulnar collateral, radial, ulnar, nutrient and muscular branches of the brachial artery.

The anterior ulnar recurrent, posterior ulnar recurrent, anterior interosseous, posterior interosseous and superficial branches of the ulnar artery.

Veins of the upper limb:

Basilic vein

Cephalic vein

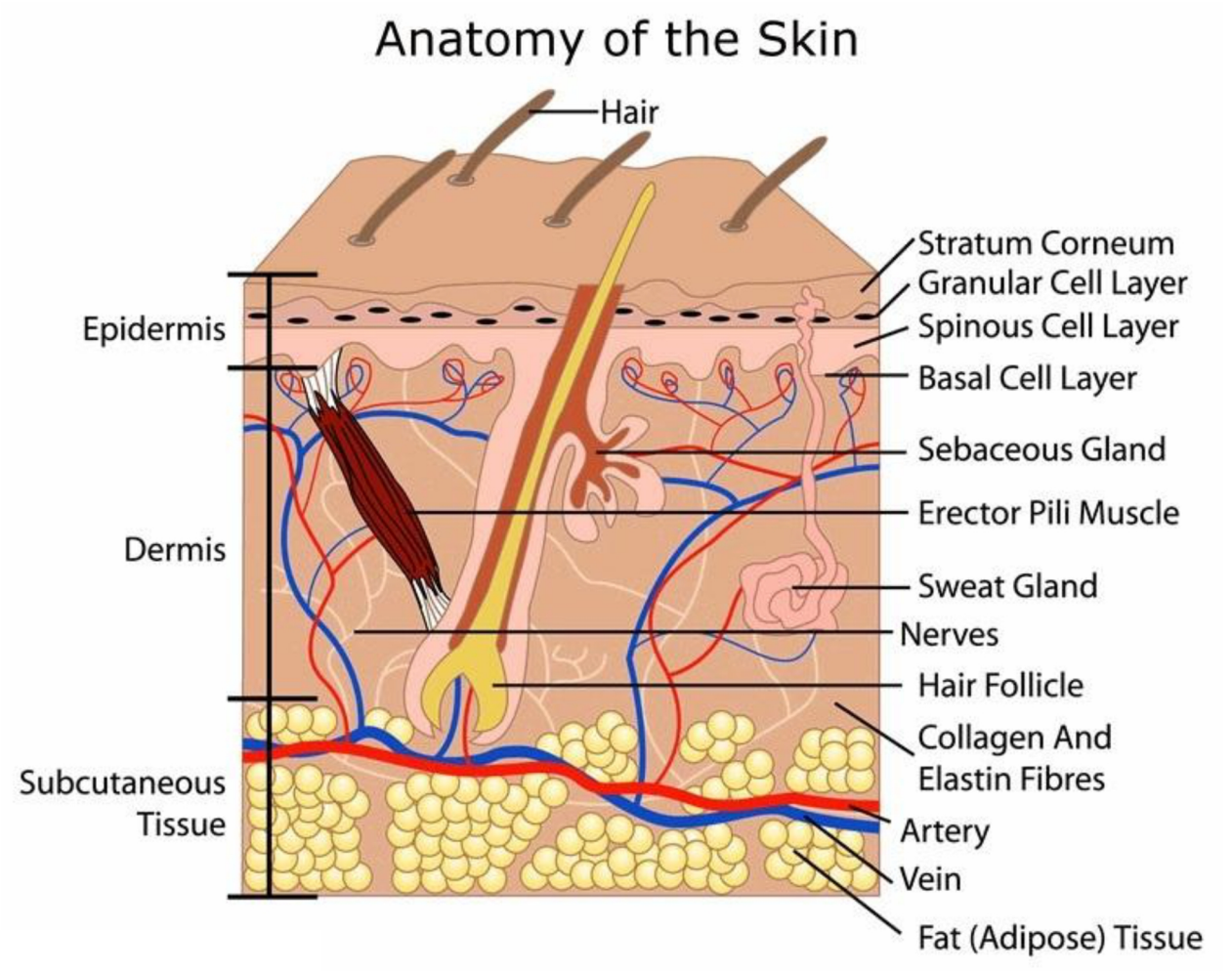
Median cubital vein

Median antebrachial vein

Dorsal venous arch

ANATOMY OF SKIN

- Skin is the complex and largest organ in our body.
- It accounts about 15 to 20 % body weight.



EPIDERMIS:

1. Stratum corneum
2. Stratum lucidum

2. Stratum granulosum

3. Stratum spinosum

4. Stratum basale

EPIDERMAL COMPONENTS: keratinocytes–cytoskeleton, langerhans cells-antigen presenting cells**,** melanocytes- produces melanin**,** merkel cells**,** epidermal appendages**,** sweat gland**,** pilosebaceous follicles.

DERMIS:

It is of two types

1.PAPILLARY DERMIS

It helps in good adhesion between epidermis and dermis, made up of loose collagen bundles and thin elastic fibre.

2.RETICULAR DERMIS.

It has thick elastic layer & coarse collagen.

Dermal layer mainly contains type 1 and type3 collagen almost about 85 to 95%. It also contains type 4 and type 7 collagen. This layer act as a mechanical barrier

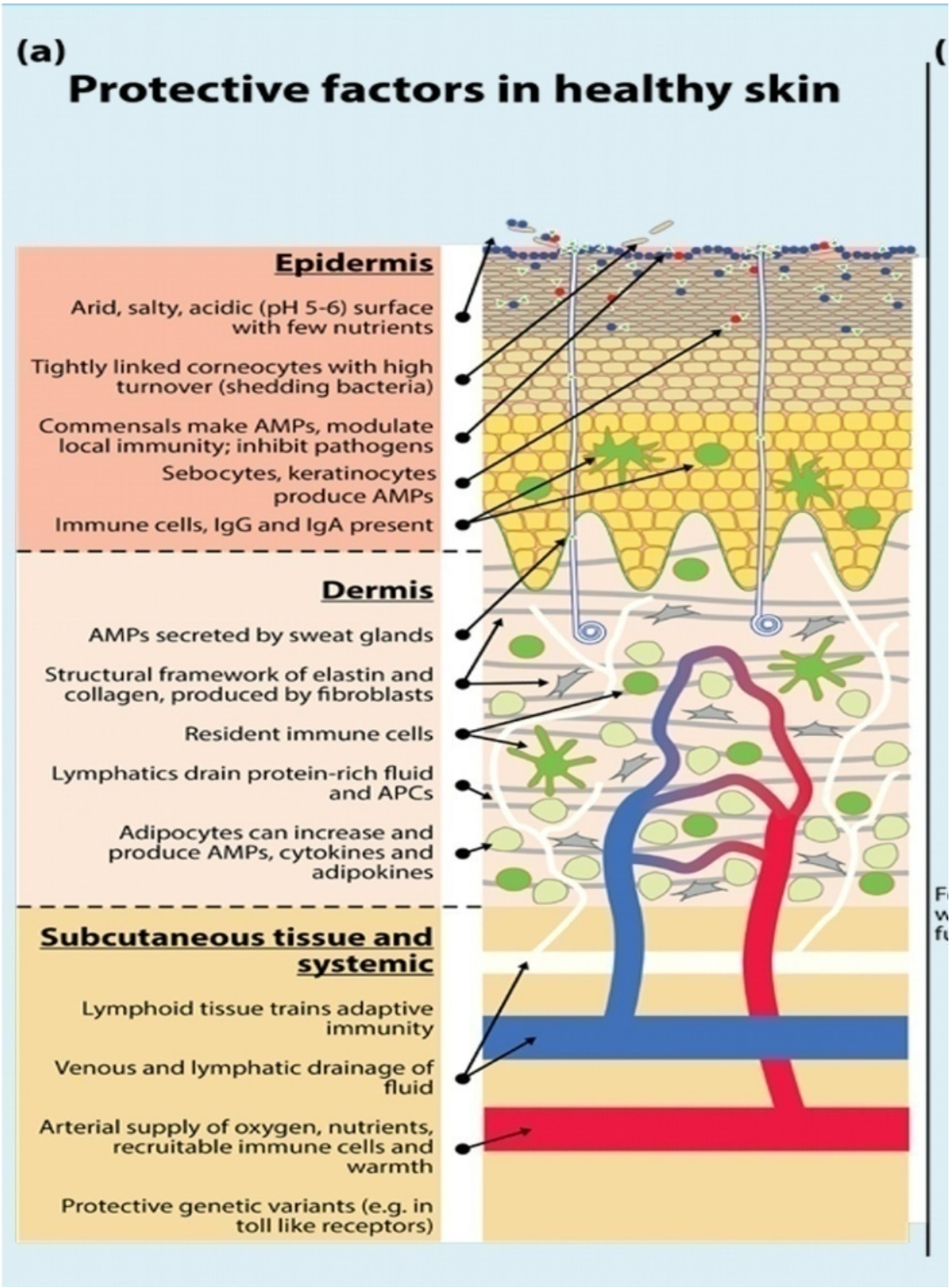
AETIOPATHOGENESIS OF CELLULITIS:

Intact skin provides protection from the external environment by serving as a physical barrier and maintaining a normal flora that is not permeable to the growth of pathogenic organisms.

The citation of Hippocrates is that cellulitis begins from skin abrasions or skin breaks is considered as risk factors from ancient period.

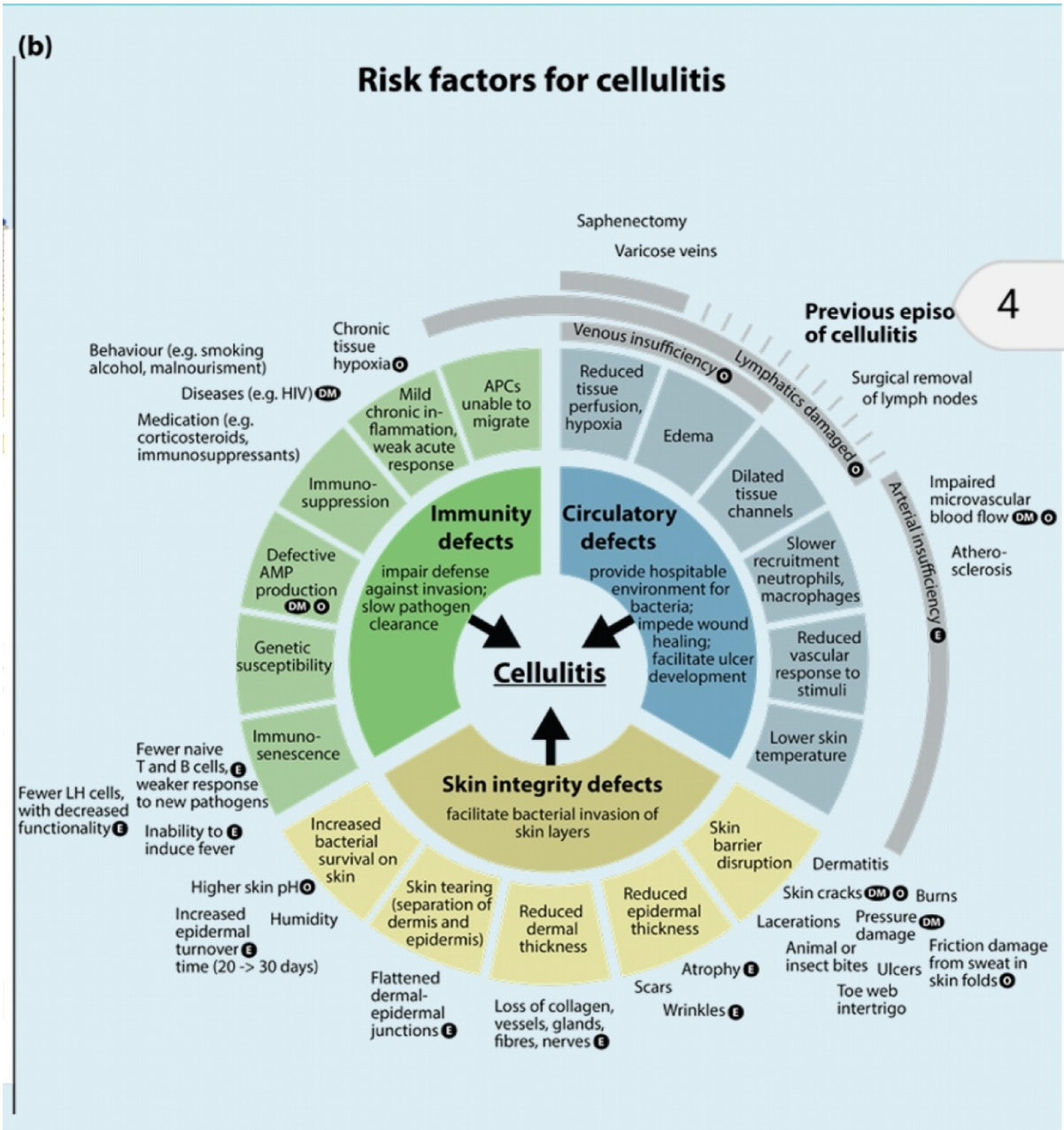
In cellulitis pathogenic microorganisms cause damage to the surrounding tissues, which leads to an inflammatory response characterized by warmth, erythema and pain.

Such damage is more complicated in patients with diabetes because long-term hyperglycemia leads to motor and autonomic neuropathy, cellular and humoral immunopathy and angiopathy.



Cellulitis occurs when an infectious organisms invades the dermis of the skin (usually through a break). This disruption of the skin can result from several causes including fungal infections (like onychomycosis, and tinea

pedis), traumatic wounds, venous leg ulcers, pressure ulcers, and web spaces infection. The natural presence of low temperature, low pH and skin flora play an important role in reducing pathogenic colonization on the skin surface.



In most of the cases, the cause for cellulitis remain unknown.

Elderly patients are at increased risk for the more severe disease[2,6].

The maceration and fungal infection of toe webs,

Known as "athletes footis considered as most important risk factor for cellulitis due to its strongassociation with cellulitis[15].

Diabetic population are prone to cellulitis. Poor glycemic allows bacteria to grow more rapidly in the affected tissue, and end up in septicemia if the infection enters the bloodstream.

The impairedimmune system and blood circulation in the legsand diabetic neuropathy in diabetes in which the ulcers may not be painful, and may get infected.

Traumatic Wounds :

Traumatic events such as cuts and bites and injection drug use result in wounds that increase the risk of cellulitis. These traumatic wounds become the site of entry of the organism and risk of skin infections and abscesses[17].

The vasculature to the skin is an important protective barrier and its breach is an important risk factors which are seen in traumatic events.

The cuts in the web space and intertrigo can cause cellulitis. Fungal infection dermatophytes lead to formation of scales and fissures forming that become the site of entry for the bacteria[3,4].

- **Infected dog and cat bite wounds[6]** are polymicrobial, with Pasteurellaspp. Streptococcus spp., Staphylococcuspp, and Moraxella spp. as the most

common aerobic organisms, and with Fusobacteriumspp., Porphyromonas, Prevotellaspp, and Bacteroidesspp. as the most common anaerobic organisms. Pasteurella spp.and Capnocytophagacanimorsun is the most common etiology of dog and cat bite infections, and many infections are caused by both aerobic and anaerobic microorganisms.

In peripheral vascular disease circulatory failure of the limb is results in arterial ulcers which is the site entry of micro organism, causing cellulitis.

In chronic venous insufficiency

Venous stasis, venous hypertension results in venous eczema which becomes the port of entry of micro organism resulting cellulitis.

It was thought that static blood within the superficial veins lead to hypoxia, which cause stasis ulcers.

At present ambulatory venous hypertension is the only accepted underlying cause of venous hypertension which is due to valve incompetence of saphenous vein, perforator veins, obstructipon of deep veins.

High venous pressure results in pericapillary infiltrates which includes fibrin and other proteins which lead to fibrosis.

Long standing edema in renal failure, congestive cardiac failure, hypoproteinemia or lymphedema,old age with immunosenescence, skin atrophy, diabetes and obesity , malnourishment,immunosuppresion and loss of

skin elasticity, lupus, vasculitis, Hansen's disease, poliomyelitis infected malignant ulcers, poor socioeconononomic status cause confer a relatively high risk of cellulitis

Based on Dupuy et al 1999, Roujeau et al. 2004, Mokni et al. 2006, Halpern et al. 2008) chronic leg edema is considered as independent riskfactors and assosciated with increased incidence of cellulitis and it is also a consequence of cellulitis.

The abnormal lymphatic absorption is seen in the patient with cellulitis and also in contralateral leg which is a predisposing factor for a recurrent cellulitis based on lymphoscintigraphy studies.

In obesity impairedbalance in lymphatic flow, i.e. overproduction or slow drainage of lymph may be involved. Several immunomediators are released from adipose tissue alter the skin barrier, micro circulation and predisposes to cellulitis and it is considered as independent risk factors.

The infection in the immune alteredpatients is always by the non-group A streptococci, In adults with intact immune system, the most common cause of cellulitis is group A streptococci **(Streptococcus pyogenes)**. Another important but less common organism is **Staphylococcus aureus**.

The recent raising concern has been about the emergence of bacteria that is resistant to antibiotics treatment, like methicillin-resistant Staph aureus(MRSA). Infections with MRSA has increased recently causing an increase in the need of the use of vancomycin, clindamycin, doxycycline and other anti-MRSA antibiotics.

Predisposing factors that increase the risk of acquiring MRSA include prisoners, homosexuals, athletes, long-term care facilities residents, previous MRSA exposure, intravenous drug abuse, and military recruits.

In atopic dermatitis and acne and perianal abscess, where more staphylococci and fewer streptococci infections are common.

The incidence of cellulitis in pediatric age group has come down due to vaccination. H.influenza type B and Streptococcus are common causes of cellulitis in the peadiatric age group.

Streptococcus pneumonia cause**malignant form of cellulitis** characterised by the tissue necrosis, suppuration,invasion into the bloodstream[10].

Mycobacterial infections[23]can present like cellulitis, in the form of subacute to chronic type and diagnosis is routinely made by the presence of langhangaint cells in the granuloma and Acid fast mycobacterial organism from the mycobacterial culture and the biopsy specimens.

DIAGNOSIS OF CELLULITIS

The typical clinical presentation of cellulitis is an acute, unilateral, spreading skin infection with poorly demarcated erythema, and typical signs of inflammation including pain, erythema, heat,swelling. Other signs include edematous lymphatics of the skin that can lead to the appearance of the orange peel sign, formation of bulla, and or inflammation of proximal lymph vessels causing lymphangitis. Tender lymphadenopathy also present in severe cases of cellulitis[4,6,11].

TYPICAL CASE OF CELLULITIS

Typical Cellulitis

The differential diagnosis include

1. Erysipelas
2. Varicose dermatitis
3. Acute lipodermatosclerosis
4. erythema nodosum.

5. pyoderma gangrenosum.

6.DVT

COMPLICATIONS :

1. Fasciitis,
2. Myositis,
3. Subcutaneous abscesses,
4. Septicaemia,
5. Gangrene
6. Post streptococcal nephritis
7. death.
8. Lymphangitis, lymphadenitis.

Differential diagnosis[24]

Infectious diseases	Clinical features resembling cellulitis	Clinical features not typical of cellulitis
Erythema migrans	Demarcated erythema	Gradual spreading of the lesion in a few days or weeks, not oedematous, only mild fever occasionally (Hytönen et al. 2008).
Necrotising infections	Ecchymosis, blisters and bullae mayoccasionally accompany cellulitis (Guberman et al. 1999)	Local pain disproportionate to skin lesion, oedema outside the erythema, patient severely ill and deteriorating, , hypotension (Anaya and Dellinger 2007)
Septic arthritis	Fever, erythema, warmth, swelling	Joint effusion, painful movementrestriction of the joint (Sharff et al. 2013)
Herpes zoster	Tingling sensation, pain, erythema	Typical clinical picture, when vesiclesappear, no fever
Primary Herpes simplex infection	Erythema, local swelling, occasionally fever	Typical vesicles, usual location in genitalarea, finger, "herpes gladiatorum"(Belongia et al. 1991)
Erysipeloid	Skin erythema with distinct border,Bullae	mild/no systemic symptoms, animalcontact (Veraldi et al. 2009)

Non-infectious conditions	Clinical features resemblingcellulitis	Clinical features not typical of cellulitis
Deep venous thrombosis	Diffuse erythema, warmth, swelling	Mild temperature rise, no fever or chills,no local adenopathy (Goodacre 2008)
Stasis dermatitis	Demarcated erythema, warmth,recurrent exacerbations	Chronic condition, often bilateral, nofever (Weingarten 2001)
Gout	Diffuse erythema, pain, recurrentAttacks	No fever, mild temperature rise possible, clinical picture often typical (Terkeltaub2003)
Systemic lupus erythematosus(lupus panniculitis)	Demarcated skin lesion, recurrent	History of systemic lupus, no systemic signs of infection (Fabbri et al. 2003)
Erythema nodosum	Raised erythematous lesions,painful, may be recurrent	Often multiple lesions, underlying infection or other cause (Psychos et al. 2000)
Contact dermatitis	Erythema, swelling, vesicles,demarcated lesion	Systemic signs absent, in chronic stateeczematous (Saint-Mezard et al. 2004)
Insect bite	Acute onset, erythema, swelling,Pain	Pruritus, systemic signs often absent,occasionally anaphylaxis (Reisman 1994)
Auricular relapsingPolychondritis	Acute inflammation, redness,warmth, swelling, tenderness,often recurrent	Occurs in cartilaginous part of ears (not in earlobe), usually bilateral, no systemic signsof infection, rare (Mathew et al. 2012)
	Indurated, annular lesion,	

Eosinophilic cellulitis (Wells syndrome)	ordiffuse erythema	Often multiple lesions in different parts ofthe body, itching, usually no fever, very rare(Wells and Smith 1979)
Neutrophilic cellulitis (Sweet's syndrome)	Fever, systemic signs, erythematous skin lesions	Usually multiple lesions, most often inupper extremities, papular or nodular(Cohen and Kurzrock 2003)
Erythromelalgia	Redness, swelling and pain inhands or feet, recurrent	Typical clinical picture, heat intolerance,cold reliefs symptoms (Norton et al. 1999)

NECROTIZING FASCIITIS[3,8]present initially with erythema, tenderness, warmth, swelling, and pain out of proportion to physical findings. As the infection progresses, blistering, skin crepitus, skin discoloration, and necrosis occur. Polymicrobial necrotizing fasciitis presents with relatively intact skin and spreads in 3–5 days. Streptococcal necrotizing fasciitis (what the lay press terms *flesh-eating bacteria*) can progress quickly within1–2 days. Clostridial gas gangrene is characterized by gas production and muscle necrosis. Diagnosis is based on clinical presentation, recent history of penetrating or blunt trauma, laboratory abnormalities, and surgical exploration. Cultures should be obtained from tissue samples and blood. Once the diagnosis is established, necrotizing fasciitis is considered a medical emergency. Immediate and aggressive surgical debridement is essential in the management of necrotizing fasciitis. Polymicrobial infections must be empirically treated with broad-spectrum antibiotics such as vancomycin, linezolid, or daptomycin plus piperacillin-tazobactam or carbapenem or ceftriaxone plus metronidazole that are directed against gram-positive cocci, Enterobacteriaceae, and anaerobes. Group A streptococcal and clostridial infections are treated with parenteral aqueous penicillin-G plus clindamycin. Adding clindamycin to β-lactam therapy provides several advantages, including additional coverage against streptococci and staphylococci, an immunomodulatory effect, and

inhibition of toxin production. Pyomyositis caused by MSSA is treated with nafcillinor oxacillin or cefazolin. The recommended total duration of therapy is 2–3 weeks. Hyperbaric oxygen therapy is not recommended because it has not been proven effective and because it may delay resuscitation and surgical debridement.

COMPARTMENT SYNDROME

Compartment syndrome is a painful condition that occurs when pressure within the muscle compartment is more than 30mmHg and this pressure can decrease blood flow, which prevents nourishment and oxygen from reaching nerve and muscle cells. It can be acute or sub acute or chronic. In acute compartment syndrome, unless the pressure is relieved quickly, permanent disability and tissue death may result. This does not usually happen in chronic (exertional) compartment syndrome[20,23]. It most commonly involves the forearm and lower leg. The cause of compartment syndrome in cellulitis is due to the circumferential cellulitis which leads on to excessive pressure on the muscle compartments.

In a normal human body, a pressure gradient is required for the blood flow from the artery to vein, blood flow from the artery to the vein is reduced when this pressure gradient is diminished. This causes excessive fluid to go out from the capillary wall into spaces between the soft tissues cells, causing

oedema and rise in intra-compartmental pressure, leading to more compression, eventually leading to lack of oxygen in the soft tissues, become hypoxic and finally tissue ischaemia and tissue death.. Measurement of intra-compartmental pressure is also important for diagnosis. A pressure higher than 30 mmHg of the diastolic pressure associated with compartment syndrome; and fasciotomy is indicated to relieve the symptoms. For those with hypotension, a pressure of 20 mmHg higher than the intra-compartmental pressure is associated with compartmental syndrome. The compartmental and the subcutaneous pressures can be measured using the special form of transducer.

GRADINGOF THECELLULITIS- BASED ON CREST CRITERIA[6]:

Class I:

No signs of toxicity and without any comorbidities

Patient can be treated in out patient department basis with oral antibiotics.

Class II

Patient may have systemic illness or not but with comorbidities like

Peripheral vascular disease Chronic venous insufficiency

Diabetes mellitus and hypertension Morbid obesity.

Class III

Patient will suffer with systemic illness, uncontrolled and unstable comorbidities and will have limb threatening complications.

Class IV

Necrotizing fasciiitis.

NECROTIZING FASCIITIS

MANAGEMENT OF CELLULITIS

Cellulitis can be diagnosed based on clinical features alone

and there is no specific investigation required for its principal diagnosis.

In Grade I cellulitis, if the patient has no risk factors and systemic signs of illness, no work up is required, can be treated as OP basis with oral antibiotics and oral analgesics[6].

In all other grades of cellulitis, all necessary investigations should be done and appropriate management should be started without any delay.

Culture and sensitivity

Sensitivity pattern of culture is generally unrewarding –

The culture in form intradermal needle aspiration, punch biopsy, swab the yield is less than 60%.

In an open wound, deeper tissue samples, collected during the time of the surgical debridement, swab taken from abscess, the yield is more than 90%.

Blood cultures is done for Class III or Class IV in case of septicaemias and it is not recommended routinely for all cases unless the patient is in septic shock.

Staphylococcus aureus and Group A streptococcus are the most common organisms cultured in the cellulitis.

Blood investigations:

- Complete blood count
- Leukocytosis, c-reactive protein and erythrocyte sedimentation rate are frequently elevated.
- Renal parameters
- Blood urea
- Serum creatinine

Class II-IV	Selected patients
- CBC - ESR/CRP - Urine routine - Culture from the break/ ulceration.	Blood cultures only in Class III or Class IV cellulitis. Streptococcal serology only in refractory cases where diagnosis is in doubt. Skin biopsy where differential diagnosis includes other non-infectious inflammatory lesions.

The arterial and venous doppler systemis done to assess the arterial flow, pattern, venous insufficiency, venous reflux and to find deep vein thrombosis.

Xray of the affected limb is done to look for the osteomyelitis changes in the bone And it may harbor themicro organism in destroyed part of bone which result in cellulitis.

Computed tomographic imaging can be taken in grade IV cellulitis to rule out the condition in patients who are stable to assess the soft tissue damage.

2)USG-guided aspiration of the pus is the newer technique has been employed now a days.

3)Magnetic resonance imaging can be a part of the work up but MRI typically takes much longer time than that of the CT scanning.

TREATMENT

In Grade I cellulitis patients is treated as outpatient basis with oral antibiotics, oral analgesics, limb elevation and patient is asked to come for regular follow.

Grade II, grade III and grade IV cellulitis patients require hospitalisation until the cellulitis and systemic signs are clinically improving, and co-morbidities are stabilised.

Patients with necrotising infection is a surgical emergency and should be dealt immediately without any delay.

Cellulitis with underneath abscess formation requires surgical drainage.

Streptococci and *S. aureus* are the most common pathogens identified in patients with cellulitis be treated with benzylpenicillin or feneticillin.

If patient is allergic to beta lactam antibiotics Co-amoxiclav and clindamycin is the alternate drug, and inhibits streptococcal and staphylococcal toxin production.

Recent study showed clindamycin is resistant to some patients as compared with flucloxacillin and hence clindamycin is preferred less as an empirical choice.

Vancomycin remains the first choice of treatment, with linezolid as an alternative in case of methicillin resistant staph aureus(MRSA).

Intravenous vancomycin with imipenem, piperacillin/tazobactam, or meropenem should also be considered in non-purulent cellulitis that presents with hypotension, high fever, immunocompromise, or sepsis are the mainstay of treatment for grade III and grade IV cellulitis.

Other novel newer antibiotics options that can cover MRSA include **tedizolid, oritavancin, telavancin, and dalbavancin**. However, these drugs are still new with no sufficient information regarding its efficacy and safety. Therefore, they are only considered in special cases.

3.1 Suitable Drug Therapy for Typical Cellulitis

	First line	Second line
Class I	Flucloxacillin 500mg qds po	Penicillin allergy: Clarithromycin 500mg bd po
Class II	Flucloxacillin 2g qds IV **or** * Ceftriaxone 1g od IV (OPAT only)	Penicillin allergy: Clarithromycin 500mg bd IV **or** Clindamycin 600mg tds IV
Class III	Flucloxacillin 2g qds IV	Penicillin allergy: Clarithromycin 500mg bd IV **or** Clindamycin 900mg tds IV
Class IV	Benzylpenicillin 2.4g 2-4 hourly IV + Ciprofloxacin 400mg bd IV + Clindamycin 900mg tds IV (If allergic to penicillin use Ciprofloxacin and Clindamycin only) **NB Discuss with local Medical Microbiology Service**	

Class II

Any one of below Empirical antibiotic is given in case of class II cellulitis.

1) IV piperacillin tazobactum 4.5g

2) IV Imipenam-cilastatin 1g 6-8 hrly

3) IV meropenem 1g 8hrly

4)IV cefotaxime 2g 6hrly

5) metronidazole 500 mg 6hrly

CLASS III

In this grade,surgical wound debridement, fasciotomy should be done in most of the cases.

Deeper tissue should be taken for culture and sensitivity. If the patient presents with signs of shock is an indication of penicillin G and clindamycin to prevent streptococcal toxic shock syndrome.

Sloughed out, dead devatilized tissue should be thoroughly debrided, and underlying abscess should be drained.

Culture is done in every two weeks and antibiotics given according to the sensitivity.

CLASS IV

Patients with necrotizing fasciitis present initially with erythema, tenderness, warmth, swelling, and pain out of proportion to physical findings. As the infection progresses,blistering, skin crepitus, skin discoloration, and necrosis occur. Diagnosis is based on clinical presentation, recent history of penetrating or blunt trauma, laboratory abnormalities, and surgical exploration.

Cultures should be obtained from tissue samples and blood. Once the diagnosis is established, necrotizing fasciitis is considered a medical

emergency (Lancerotto 2012). Immediate and aggressive surgical debridement is essential in the management of necrotizing fasciitis.

Polymicrobial infections must be empirically treated with broad-spectrum antibiotics such as vancomycin, linezolid, or daptomycin plus piperacillin-tazobactam or carbapenem or ceftriaxone plus metronidazole that are

directed against gram-positive cocci, Enterobacteriaceae and anaerobes. Group A streptococcal and clostridial infections are treated with parenteral aqueous penicillin G plus clindamycin. Adding clindamycin to β-lactam therapy provides several advantages, including additional coverage against streptococci and staphylococci, an immunomodulatory effect, and inhibition of toxin production. Pyomyositis caused by MSSA is treated with nafcillin or oxacillin or cefazolin.

The recommended total duration of therapy is 2–3 weeks. Hyperbaric oxygen therapy is not recommended because it has not been proved effective and because it may delay resuscitation and surgicaldebridement.

Bernard et al. 1992, conducted a randomised, open multicenter trail comparing the effectiveness of Roxithromycin per oral with Penicillin Intravenous among 69 hospitalized patients diagnosed to have cellulitis. Thestudy results showed that cure without additional antibiotics with use of roxithromycin is (84%) and that with penicillin is (76%) (P = 0.43)

Bernard et al.2002, in one of his Randomised, non-inferiority, open, multicenter study compared the effectiveness of Pristinamycin per oral with penicillin intravenous and then per oral in 289 hospitalised adults with diagnosis of cellulitis. Results showed that Intention to treat: cure at follow-up pristinamycin is (65%) and that for penicillin is 79/150 (53%).

Grayson et al. 2002, in his Randomized, double-blind equivalent trial compared the effectiveness of Cefazolin intravenous + probenecid with ceftriaxone intravenous + placebo in 134 adult patients with moderate to severe cellulitis. Results showed that clinical cure at one month with cefazolin- probenecid found to be 46/56 (82%) and that with ceftriaxone-placebo was found to be 50/57 (85%), p=0.55

Similarly, Pallin et al. 2013 in his Randomized, placebo-controlled,double-blind, multicenter study compared the effectiveness of Cephalexin +Trimethoprim-Sulphamethoxazole with cephalexin + placebo in around 153 outpatients with age ≥12 months, diagnosed to have cellulitis. Results showed that cure when adding Trimethoprim-Sulphamethoxazole with cephalexin were found to be 62/73 (85%) and that with controls it showed 60/73 (82%).

Likewise Hepburn et al 2004, in his Randomized, placebo-controlled,double-blind, single centre study compared the effectiveness of Levofloxacin given10 days with that of Levofloxacin given for 5 days followed by placebo 5 days in around 82 patients with cellulitis as diagnosis. Results showed that the cure at 28 days with levofloxacin 10 d was found to be 42/43 (98%) and that of levofloxacin 5 d was found to be 43/44 (98%).

Zeglaoui et al. 2004, in their Randomised, open, single centre studycompared the effectiveness of Penicillin intramuscular with penicillin intravenous use in 112 hospitalised adult patients diagnosed to have cellulitis. Results showed Failure rate with penicillin intravenous 20% and that of penicillin intramuscular 14% , with p=0.40

METHODOLOGY OF STUDY

100 patients who got admitted for cellulitis and its complications, willing for regular follow up after discharge who got admitted under all surgical units of Tirunelveli Medical College Hospital, were included as the study group.

STUDY DESIGN: Prospective study

SOURCE OF DATA: 100 patients of CELLULITIS getting admitted in surgical ward

STUDY PERIOD: August 2017 to February 2018

INCLUSION CRITERIA:

1. Patients with cellulitis of lower limb and upperlimb aged above 12 years of age.

2. Cellulitis of lower limb and upper limb presenting with or without abscess and ulcers.

EXCLUSION CRITERIA:

1. Patients less than 12years of age**.**

MATERIALS:

Age incidence,Sex incidence,Limb involved,Severity during presentation.

Basic blood investigations.

Swab and deeper tissue biopsy - culture from the wound.

The sensitivity pattern of the organisms cultured.

Arterial and venous pathology by Doppler study and x ray for the osteomyelitic changes in bones of cellulitis.

METHODOLOGY

All the 100 patients presenting with features of cellulitis whose diagnosis were made by clinical findings admitted in the general surgery ward. Details of the patient were noted by detailed history regarding the presenting illness, pain, reddening of the region, swelling of the local part, any ulcerations, blister/ bleb formation, history of any trauma, unknown bites including the comorbid conditions , smoking and alcoholic history.

In the clinical examination, general examination of the patient for the presence of the anemia and jaundice ,nutritional and hydration status of the patient has been recorded.

Systemic examination of the cardiac system, respiratory system, abdominal examination and central nervous system have also examined.

Vital parameters- pulse rate, blood pressure, respiratory rate and temperature

Local examination of the lower limb,

Extent of cellulitis, Presence of subcutaneous abscesses, blisters/blebs, ulcers, serous or purulent discharge.

Distal pulsations and capillary refill time was assessed.

Careful assessment for complications like compartment syndrome, fasciitis, myositis, subcutaneous abscess, septicaemia.

Following clinical examination, routine investigations , Doppler study of the affected limb and X-ray of the affected limb was taken to rule out osteomyelitis. Treatment were started as soon as diagnosis is made by intravenous fluids, iv antibiotics and wound debridement. And bacteriological cause is identified by culture which is done for both aerobic and anaerobic bacteria.

The samples were then cultured in blood agar and Mac conkey agar for aerobic bacteria and Robertson's cooked meat media for anaerobic bacteria.

Following initial debridement the wound was inspected daily and surgical wound debridement done periodically and thorough wash given by povidone iodine, hydrogen peroxide, EUSOL bath and normal saline. Once the granulation occurs and wound is managed with secondary suturing or split skin grafting.

GRADING OF CELLULITIS AS DEVISED BY ERON

2.1 Clinical Classes of Cellulitis

A classification system can serve as a useful guide to admission and treatment decisions. This classification was devised by Eron [4] for skin and soft tissue infections.

Class I patients have no signs of systemic toxicity, have no uncontrolled co-morbidities and can usually be managed with oral antimicrobials on an outpatient basis.

Class II patients are either systemically ill or systemically well but with a co-morbidity such as peripheral vascular disease, chronic venous insufficiency or morbid obesity which may complicate or delay resolution of their infection.

Class III patients may have a significant systemic upset such as acute confusion, tachycardia, tachypnoea, hypotension or may have unstable co-morbidities that may interfere with a response to therapy or have a limb threatening infection due to vascular compromise.

Class IV patients have sepsis syndrome or severe life threatening infection such as necrotizing fasciitis.

LABORATORY INVESTIGATIONS:

Complete blood count:

The haemoglobin of the individuals and the Differential count, which has the impact in the clinical improvement of the patient.

FASTING BLOOD SUGAR: Fasting blood sugar more than 120mg% is indicative of diabetes.

*POST PRANDIAL BLOOD SUGAR :*After two hours of food intake,

if it exceeds 180 mg % is indicative of diabetes mellitus.

Blood Urea and serum creatinine are done to assess the renal functions of patients like chronic kidney disease, for hydration status of the patient during the course of management had the major impact on the outcome

of the patients, and it can alarm the possibility of acute renal shutdown during the management of the cellulitis.

Other blood investigations include liver function tests coagulation profile, serum protein, serum electrolytes, and HIV and HbSAg assays.

Culture and sensitivity:

Pus from infected area is cultured for microorganism of both aerobic and anerobicand their sensitivity to various antibiotics is assesed so that appropriate antibiotic can be administered to control the infection.

Doppler study of arterial and venous system:

Arterial Doppler helps in assessing the alterations in the arterial flow pattern and Venous Doppler helps in identifying the venous reflux and venous stasis ulcer,to rule out deep venous thrombosis.

Plain X-ray of the limb involved

To rule out the cases of osteomyelitis and underlying bone involvement in severe forms of cellulitis which is the site of entry of infections..

TREATMENT

Empirical antimicrobial therapy can be started for the infections caused by the staph aureus and streptococcal infections which is the common organism causing cellulitis.

Flucloxacillin is administered for the Class I infections and intravenously for Class II and Class III infections as it exerts a bactericidal effect on streptococci as well as staphylococci.

The combination of benzylpenicillin and flucloxacillin has been proven efficacy and it is administered regularly in hospitalized patients.

Patient is monitored regularly during the course of treatment for clinical improvement for seven days with same antibiotics and if the disease progress patient is started with newer antibiotics as per the sensitivity pattern for the organism grown in culture.

SURGICAL INTERVENTION:

Cellulitis with skin blistering either deroofing or aspiration of the blisters can be done and sometimes spontaneous rupture of the blisters can occur.

In cases of advanced cellulitis, the dead devatalizied tissues are excised and thorough wound wash with antiseptic solutions, tissue biopsy are taken for culture sensitivity and daily regular surgical debridement is done until the granulation occurs.

Fasciotomy was done when there was a tension and swelling at the site of cellulitis and threat of compartment syndrome.

The wounds were thoroughly washed with povidone iodine,hydrogen peroxide, well irrigated with normal saline and hemostasis attained.

Following initial debridement the wound was inspected daily and surgical wound debridement done periodically for granulation to occur and to manage with secondary suturing or split skin grafting.

The patients with cellulitis due to snake bites, arterial and venous ulcers and few patients with trauma with gangrenous changes and bony destruction and amputation was done at appropriate level necessary to remove the persisting infection.

Patients with life-threatening necrotizing cellulitis, with distal decreased blood flow with the threat of impending sepsis syndrome, were taken for amputation at appropriate levels.

The outcome of the patient and management has been analysed and recorded as whether the patient had an uneventful recovery or developed a raw area which needed further management, or the patient had a residual deformity or the patient had died because of the disease.

The resultant raw area was managed with either delayed primary closure or allowed to heal by the secondary intention. split thickness skin grafting has been done.

LIMITATIONS:

Cellulitis of grade I are treated as OP basis with oral drugs and asked to come for regular follow up. Hence this study principally being done to hospitalized patients of higher grades of cellulits.

The incidence of anerobic infections could not be studied as the culture facilities to grow anaerobic organism is not available.

RESULTS & OBSERVATIONS

The findings observed in my study conducted in Tirunelveli Medical College regarding the management of lower limb cellulitis of 100 are listed as follows.

1. AGE DISTRIBUTION

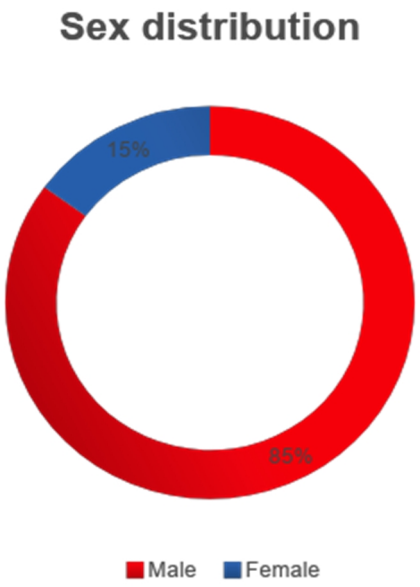
1. AGE DISTRIBUTION

Sl.No.	Age Group	No of cases
1.	<20	1
2.	21 – 30	5
3.	31 – 40	16
4.	41 – 50	30
5.	51 – 60	34
6.	>60	14

Out of 100 patients studied 64 patients were belonging to the age group of 41-60 and it is evident that the incidence of cellulitis increases as the age increases.

2. SEX DISTRIBUTION

2. SEX DISTRIBUTION	
Male	Female
85	15



In my study of 100 patients the incidence of cellulitis common among the male individuals than female with 15%.

3. LIMB INVOLVED

Out of 100 patients 76 patients had lower limb cellulitis and 24 patients had upper limb cellulitis in my study.

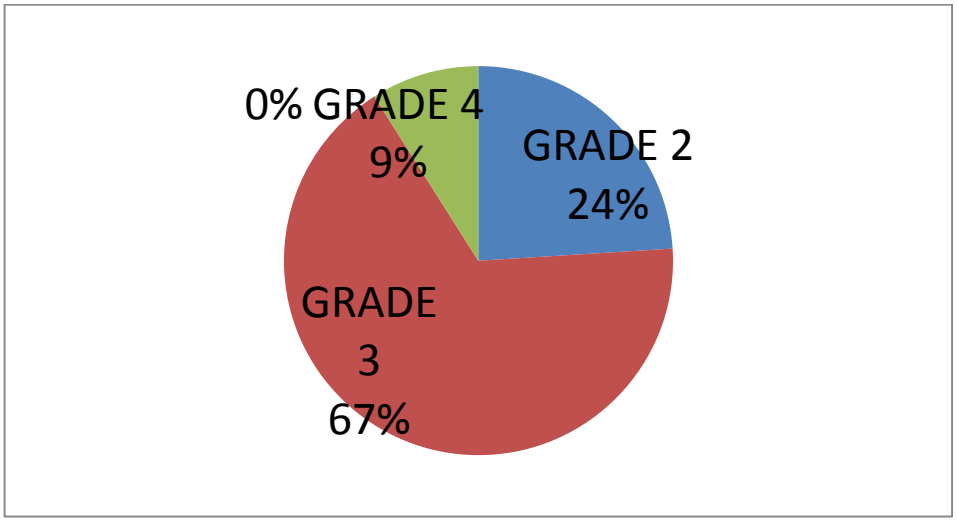
upper limb	24
lower limb	76

4. GRADE OF CELLULITIS:

Cellulitis has been graded based on CREST criteria.

TABLE: 4 GRADE OF CELLULITIS

Sl.No.	Grade	No. of cases
1.	II	24
2.	III	67
3.	IV	9



67 individuals belong to the grade III cellulitis, whereas 24 patients grade II and 9 patients belongs to IV respectively out of 100 patients studied with incidence of higher grades of cellulitis are being admitted and treated.

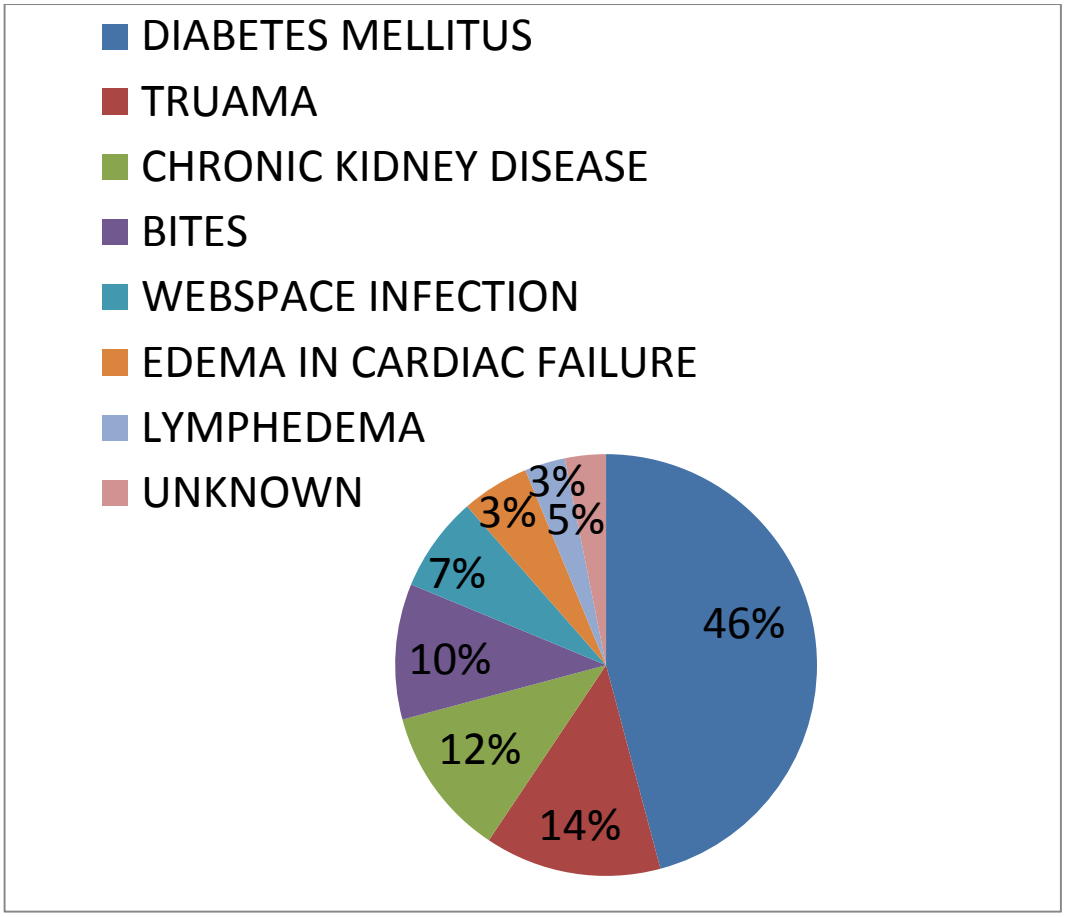
5. THE CAUSE OF CELLULITIS

The causes for the cellulitis in the study group such as diabetes mellitus, bites, infected venous ulcers, web space infections, infected traumatic ulcers, cellulitis imposing on theedematousand lymphedematous limb and in the patientsof the renal and cardiac failure patients are tabled below.

In few patients exact cause of the cellulitis is not diagnosed.

TABLE 5: CAUSE OF CELLULITIS

Cause	No. of Patients
Diabetes mellitus	46
Infected traumatic wounds	14
Chronic kidney disease	12
Bites	10
Web space infections	7
Edema in cardiac failure	5
Lymphedema	3
Unknown	3



The diabetes mellitus is responsible for most cases of cellulitis in the study group. cellulitis seen in chronic kidney disease, lymphedema and cardiac failure constitutes a low proportion as the etiology. The cause of cellulitis is unknown in 3% of my study group.

6. MICRO-ORGANISMS CULTURED

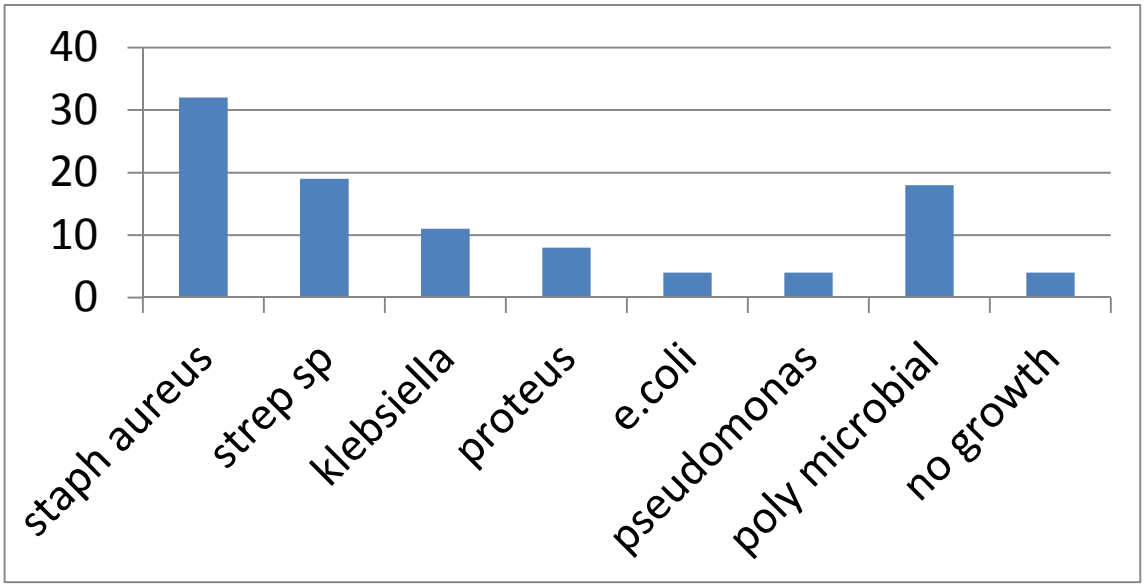
78 patients had monomicrobial infections, the infection is poly-microbial in 18 patients and no growth in culture has been observed in 4 individuals.

The individual organisms cultured is tabled below

TABLE: 6 MICRO-ORGANISMS CULTURED

Sl.No.	Organisms	No. of Patients
1.	Staphylococcus aureus	32
2.	Streptococcus SP	19
3.	Klebsiella SP	11
4.	Proteus SP	8
5.	E-Coli	4
6.	Pseudomonas SP	4
7.	Polymicrobial	18
8.	No Growth	4

The results are charted as follows.



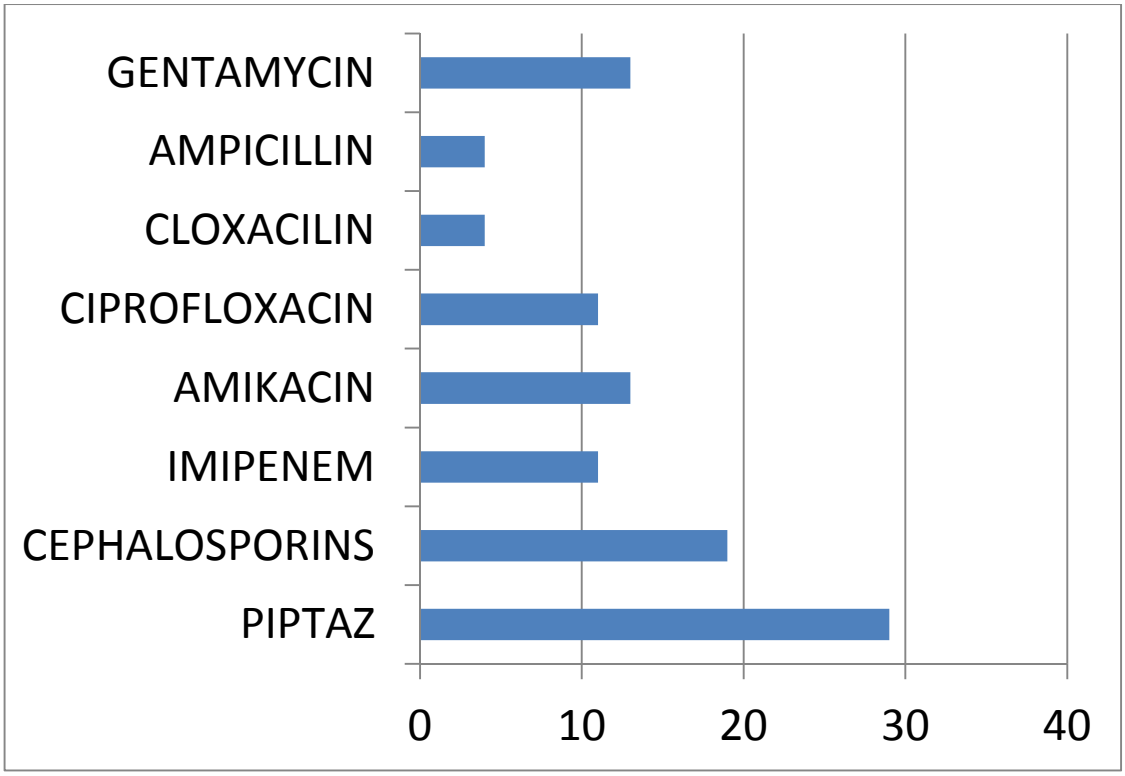
Staphylococcus and streptococcus species were the common organisms responsible for the cellulitis and other organisms include klebsicllaSP, proteusSP, pseudomonas SP, Ecoil also contribute in the considerable proportion in causing infections.

7. SENSITIVE DRUGS

Culture positivity were found in 96 patients among 100 patients observed. The result of sensitivity pattern for the organisms is listed below.

Sl.No.	Antibiotic	No. of Patients
1.	Piperacillin – Tazobactum	29
2.	Cephalosporin group	19
3.	Imipenam	11
4.	Amikacin	13
5.	Ciprofloxacin	11
6.	Cloxacillin	4
7.	Ampicillin	4
8.	Gentamycin	13

The piperacillin – Tazobactum and imipenam are the two group of antibiotics to have the maximum sensitivity for the cellulitis causing common organism and the results are plotted as follows.


GENTAMYCIN
AMPICILLIN
CLOXACILIN
CIPROFLOXACIN
AMIKACIN
IMIPENEM
CEPHALOSPORINS
PIPTAZ
0
10
20
30
40

8. CIRCULATORY CHANGES OBSERVED

The arterial Doppler and venous Doppler done to the patients in our study group had no DVT and monophasic flow pattern seen in 14 patients and no flow pattern in 1 patients and normal triphasic flow pattern in 85 individuals.

Sl.No.	Changes observed	No. of Patients
1.	No flow in calf vessels	1
2.	Monophasic flow in peroneal artery	4
3.	Monophasic flow in posterior tibial artery	6
4.	Venous insufficiency	4
5.	Deep vein thrombosis	0

9. BONE INVOLVEMENT

Out of 100 patients 13 patients showed osteolytic changes which required amputation among the patients with risk factors of diabetes mellitus, snake bites at site of bite of the toes or the metatarsal below showed lytic changes, or destruction due to the gangrenous changes and no other bony changes were observed in the patients.

10.TREATMENT

Treatment of the individuals varied according to the severity of the disease, some patients were managed conservatively with parenteral antibiotics, the anti-inflammatory agents and limb elevation so as to reduce the associated edema, while majority of the others required surgical wound debridement with or without decompression of the fascial compartment by a fasciotomy. Very few patients needed amputation of the limb.

Table No 10

Sl.No.	Management	No. of Cases
1.	Conservative	13
2.	Wound debridement	27
3.	Wound with debridement fasciotomy	48
4.	Amputation	12

It is observed that around 75 patients in the study group required surgical debridement, 48 of them required decompression of the muscular compartment by means of a fasciotomy. Around 12% of individuals in the study group required amputation.

11.CONSERVATIVE MANAGEMENT

GRADES	UPPER LIMB	LOWER LIMB
I	6	7
II	-	-
III	-	-

It is observed that 6 patients of grade I cellulitis out of 24 upper limb patients and7 patients of grade II cellulitis out of 76 lower limb patients managed conservatively.

12.FASCIOTOMY

GRADES	UPPER LIMB	LOWER LIMB
II	-	9
III	10	29
IV	-	-

It is observed that 9 and 29 patients of grade II and grade III cellulitis out of 76 lower limb patients and 10 patients of grade III cellulitis out of 24 upper limb patients required fasciotomy.

13.WOUND DEBRIDEMENT

GRADES	UPPER LIMB	LOWER LIMB
II	-	2
III	8	9
IV	-	8

It is observed that 8 patients of grade III cellulitis out of 24 upper limb patients and 2 patients of grade II, 9 patients of gradeIII, 8 patients of grade IV cellulitis out of 76 lower limb patients required wound debridement.

14.AMPUTATION

GRADES	UPPER LIMB	LOWER LIMB
II	-	-
III	-	11
IV	-	1

It is observed that 12 and 1 patients of grade III and grade IV cellulitis out of 76 lower limb patients required amputations.

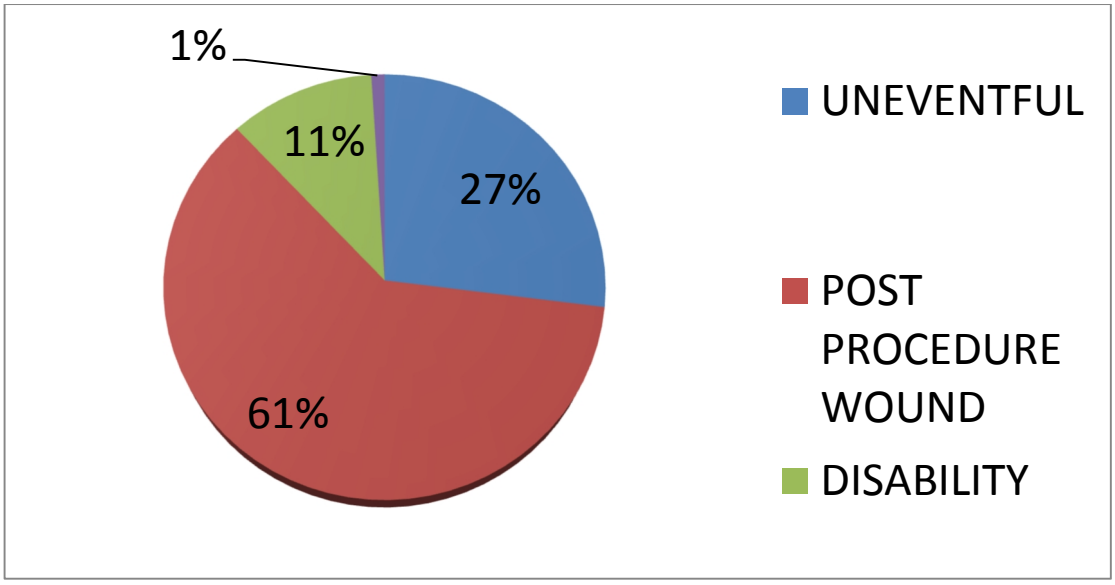
15.OUTCOME

The outcome of the treatment done has been studied, whether it is universal, or whether patient remained with a wound that needs, further managements or patient had some residual deformity or the patient had expired because of the comorbidities complicating the disease.

TABLE: 11 OUTCOME

Sl.No.	Outcome	No. of cases
1.	Uneventful	27
2.	Post procedure wound	61
3.	Disability	11
4.	Death	1

It can be seen from the table that almost all the patients managed conservatively, had an uneventful recovery and among those needed surgical intervention, 61 patients had the residual wound that needed further attention, 11 patients remained with disability amputations being done and around 1 patient died because of the disease.



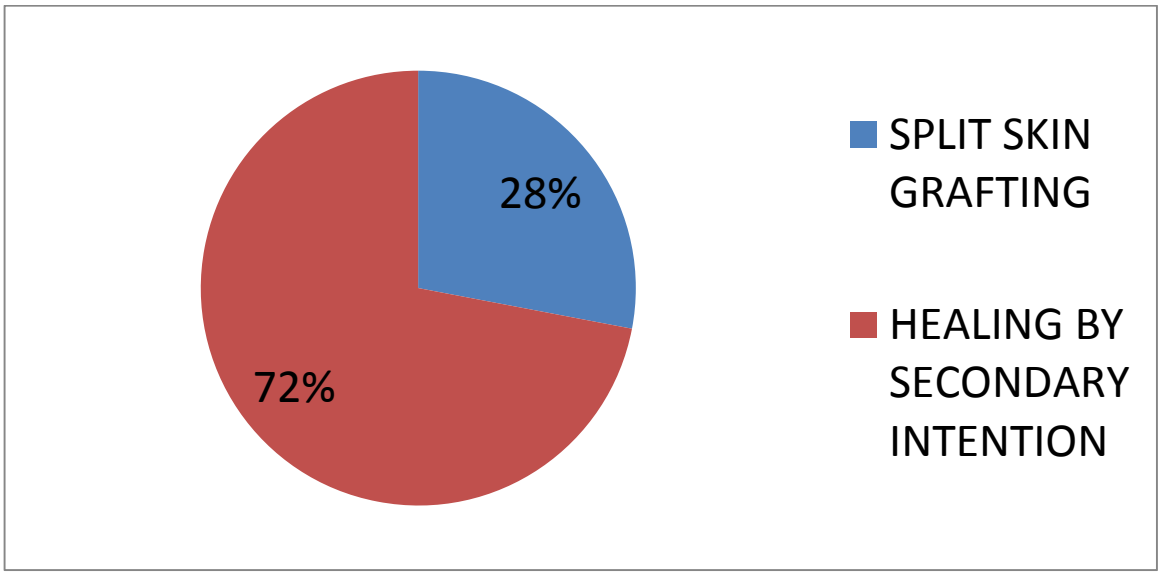
This pie chart shows the graphical representation of the observations above. This indicates about the load of patients with residual wound who need further care for the same.

12.MANAGEMENT OF THE WOUND

TABLE:11 MANAGEMENT OF THE WOUND

Sl. No.	Management of the wound	No of cases	Percentage
1.	Split skin grafting	17	28
2.	Healing by secondary intention	44	72

61 patients in the study group with resultant wound were managed ultimately with either a split thickness or allowed to heal by secondary intention.



It can be observed that from the table and graphical representation that around 28% of the resultant wounds were managed with split skin grafting and remaining 72% of the wounds healed by secondary intention.

DISCUSSION

100 patients were included in my prospective study who got admitted for cellulitis and its complications, in Tirunelveli Medical College Hospital under general surgical unit in the period of one and half years and the results observed from the study are discussed below.

Out of 100 patients studied 64 patients were belonging to the age group of 41-60 and it is evident that the incidence and severity of cellulitis increases as the age increases which correlates with literature due to immunocompromised state and associated comorbidities.

The diabetes mellitus, congestive cardiac failure and renal failures with long standing edema are common risk factors and minor contribution from immunocompromised patients, old age and obesity can cause cellulitis.

Higher grades of cellulitis in lower limb and with bilateral involvement of lower limb in chronic long standing edema are the common presentation which correlates with study by Dupuy et al 1999, Roujeau et al. 2004, Mokni et al. 2006, Halpern et al. 2008.

In my study of 100 patients the incidence of cellulitis common among the male individuals and female with 15%.

The most early forms of grade 1 cellulitis are managed on the outpatient basis with oral analgesics, oral antibiotics and to follow up regularly.

The lower limb presentation is more common and with high incidence than the upperlimb as the breach in the skin is high in lowerlimb because of bare foot walking and poor hygiene and 3 patients had unknown etiology. The people near by Tirunelveli have agriculture as their occupation and are used to bare foot walking, prone to trauma to the foot insect//snake/unknown bites in lowerlimb than upperlimb causing cellulitis, the result which correlates with literature.

Culture positivity were found in 96 patients among 100 patients observed. 78 patients had monomicrobial infections, the infection is poly-microbial in 18 patients and no growth in culture has been observed in 4 individuals.

Staphylococcus and streptococcus species were the common organisms causing the cellulitis in my study which correlates with literature and other organisms include klebsiella SP, proteus SP, pseudomonas SP, Ecoli also contribute in the considerable proportion in causing infections.

Piperacillin tazobactam and imipenem, cephalosporin group of antibiotics, amikacin, ciprofloxacin and gentamycin are the antibiotics sensitivity to most of the microorganism cultured and piptaz, imipenem are the

antibiotics with maximum sensitivity. Comparing from the literature from my study, it is evident that resistance to common antibiotics which we routinely follow have started evolving.

The arterial Doppler and venous Doppler done to the patients in our study group had no DVT and monophasic flow pattern seen in 14 patients and 1 patient had no flow pattern and normal triphasic flow in majority of individuals and venous insufficiency in 4% of individuals.

Out of 100 patients 13 patients required amputation among the patients who have osteolytic changes in the bone.

It is observed that majority of patients required surgical management such as the wound debridement, fasciotomy, ray's amputation as most of the patients presenting with higher grades of cellulits.

It is observed from study that 6 patients of grade I cellulitis out of 24 upper limb patients and 7 patients of grade II cellulitis out of 76 lower limb patients managed conservatively.

And 9 and 29 patients of grade II and grade III cellulitis out of 76 lower limb patients and 10 patients of grade III cellulitis out of 24 upper limb patients were managed by fasciotomy

And 8 patients of grade III cellulitis out of 24 upper limb patients and 2 patients of grade II, 9 patients of gradeIII, 8 patients of grade IV

cellulitis out of 76 lower limb patients were managed by wound debridement.

And 11 of grade III and 1 of grade IV cellulitis out of 76lowerlimb patients were amputated.

It is observed that almost all the patients managed conservatively, had an uneventful recovery and among those needed surgical intervention, 61 patients had the residual wound that needed further attention, 11 patients remained with disability amputation being done and around 1 patient died because of the disease.

It is analyzed that among 61 patients with residual areas ,28% of patients were managed with split skin grafting and remaining 72% of the wounds healed by secondary intention.

CONCLUSION

The incidence of cellulitis common among the age group of 40-60 years.

Males are more susceptible for cellullitis than females populations.

Higher grades of cellulitis are more common presentation in my study.

The diabetes mellitus is responsible for most cases of cellulitis in the study group followed by the traumatic ulcers which have been infected and post bite cellulitis. Cellulitis seen in chronic kidney disease, lymphedema and cardiac failure constitutes a low proportion as the etiology. The cause of cellulitis is unknown in 3% of my study group.

Staphylococcus species and streptococcus species are the common organisms responsible for the cellulitis in the study group.

The piperacillin tazobactum and imipenam were the two groups of antibiotics which tend to have the maximum sensitivity for the common organisms causing the cellulitis.

The arterial Doppler showed normal triphasic flow pattern in 85 individuals and monophasic flow pattern in 14% individuals.

Venous insufficiency has been noticed in 4% of individuals.

Around 12% of individuals in the study group required amputation.

It is observed from study that 6 patients of grade I cellulitis out of 24 upper limb patients and 7 patients of grade II cellulitis out of 76 lower limb patients managed conservatively.

75 patients had surgical debridement, 48 had decompression of the muscular compartment by means of a fasciotomy.

Around 12% of individuals in the study group required amputation.

due to osteolyticchanges and below knee amputation done in one patient.

61 patients had the residual wound that needed further attention, 11 patients remained with disability and around 1 patient died because of the septicemia.

In 61 patients with residual areas ,17 of patients were managed with split skin grafting and remaining 44 patients wounds healed by secondary intention.

This study on cellulitis found that diabetes mellitus is the most common cause followed by traumatic infected ulcer, post bite cellulitis, chronic kidney disease.

Hence early diabetes mellitus screening and good glycaemic control prevent the incidence of cellulitis.

Educating the people regarding proper foot care, foot wear usage can prevent cellulitis occurring due to web space infections, cracks in the sole, trivial trauma in the foot.

Hospital admission for the severe forms of cellulitis, appropriate and emergency surgical intervention as needed, employing culture directed antibiotics, managing the comorbidities can salvage the limbs and lives.

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PROFORMA

Name : Age :

Sex : Male ☐ Female ☐

Occupation :

Limb involved : upper limb ☐ lower limb ☐

Symptoms :

Pain ☐ Reddening ☐ Swelling ☐ Ulcers ☐ Bleb ☐ Blisters ☐

Comorbid illness :

Hypertension ☐ Epileptic ☐ Diabetes Mellitus ☐ CKD ☐

CAHD ☐ COPD ☐

Previous history of cellulitis:

Smoker ☐ Tobacco user ☐ Alcoholic ☐

Cause :

General Examination

Nourishment : Hydration status :

Pallor : Icterus :

CVS: RS: P/A:

Vital parameters

Pulse : BP :

Temperature : Respiratory rate :

Examination of limbs:

Extent of cellulitis : Blisters/blebs :

Subcutaneous abscess : Ulceration :

Discharge : Healing :

Deeper tissue involvement: Web space infection:

Breaks/ Cracks : Distal pulsations :

Colour of the limb : Sensation :

Motor activity : Others :

Lab investigations :

Complete Blood Count

Hb% : RBC :

Differntial count : Platelet :

ESR :

Blood Sugar

Fasting : B. Urea :

Post-prandial : S. Creatinine:

Culture and sensitivity :

(From the wound either by swab / tissue biopsy)

Liver function test :

Coagulation profile :

HIV ELISA :

HbS Ag serology :

Doppler study of the arterial and venous system :

Plain X-ray of the limb :

Treatment :

Conservative ☐

Surgical
- Wound debridement ☐
- Wound debridement & Fasciotomy ☐
- Amputation ☐

Outcome :

- Uneventful ☐
- Wound to be managed ☐
- Disability ☐
- Death ☐

Management of the wound:

Split skin grafting ☐ Delayed primary suturing ☐

Healing by secondary intention ☐

ABBREVIATIONS

Limb Involvement

UL	-	Upper limb
LL	-	Lower Limb

Causes:

B	-	Bites
DM	-	Diabetes Mellitus
T	-	Trauma
V	-	Venous ulcer
IN	-	Intertrigo / Web Space Infections
RF	-	Renal failure
LY	-	Lymphedema
Un	-	Unknown Cause

Organisms

St	-	Streptococcus sp.
S	-	Staphylococcus aureus
K	-	Klebsiella
Pr	-	Proteus sp.
Ps	-	Pseudomonas Aeruginosa
E	-	E.coli
NG	-	No growth

Sensitive drugs

P	-	Piperacillin-tazobactum
I	-	Imipenam
Ami	-	Amikacin
G	-	Gentamycin
Cip	-	Ciprofloxacin
Amp	-	Ampicillin
Clox	-	Cloxacillin

In the Doppler study

MF	-	Monophasic flow
NF	-	No flow
PT	-	Posterior tibial artery
P	-	Peroneal
VI	-	Venous insufficiency

X ray-limb

N	-	Normal study

Regarding management, (Rx)

WD	-	Wound debridement
F	-	Fasciotomy
Con	-	Conservative
BK amp	-	Below knee amputation

Regarding the outcome,

W	-	Resultant wound
Un	-	Uneventful
D	-	Disability
SSG	-	Split skin grafting
HS	-	Healing by secondary intention

நோயாளிகளுக்கு அறிவிப்பு மற்றும் ஒப்புதல் படிவம்
(மருத்துவ ஆய்வில் பங்கேற்பதற்கு)

ஆய்வு செய்யப்படும் தலைப்பு:
பங்கு பெறுவரின் பெயர்:
பங்கு பெறுவரின் வயது:

		பங்கு பெறுவர் இதனை ✓ குறிக்கவும்
1.	நான் மேலே குறிப்பிட்டுள்ள மருத்துவ ஆய்வின் விவரங்களை படித்து புரிந்து கொண்டேன். என்னுடைய சந்தேகங்களை கேட்கவும், அதற்கான தகுந்த விளக்கங்களை பெறவும் வாய்ப்பளிக்கப்பட்டுள்ளது என அறிந்து கொண்டேன்.	☐
2.	நான் இவ்வாய்வில் தன்னிச்சையாக தான் பங்கேற்கிறேன். எந்த காரணத்தினாலோ எந்த கட்டத்திலும், எந்த சட்ட சிக்கலுக்கும் உட்படாமல் நான் இவ்வாய்வில் இருந்து விலகி கொள்ளலாம் என்றும் அறிந்து கொண்டேன்.	☐
3.	இந்த ஆய்வு சம்பந்தமாகவோ, இதை சார்ந்து மேலும் ஆய்வு மேற்காள்ளும் போதும் இந்த ஆய்வில் பங்குபெறும் மருத்துவர் என்னுடைய மருத்துவ அறிக்கைகளை பார்ப்பதற்கு என் அனுமதி தேவையில்லை என அறிந்து கொள்கிறேன். நான் ஆய்வில் இருந்து விலகிக் கொண்டாலும் இது பொருந்தும் என அறிகிறேன்.	☐
4.	இந்த ஆய்வின் மூலம் கிடைக்கும் தகவலையோ, முடிவையோ பயன்படுத்திக் கொள்ள மறுக்க மாட்டேன்.	☐
5.	இந்த ஆய்வில் பங்கு கொள்ள ஒப்புக் கொள்கிறேன் எனக்கு கொடுக்கப்பட்டஅறிவுரைகளின் படி நடந்து கொள்வதுடன், ஆய்வை மேற்கொள்ளும் மருத்துவ அணிக்கு உண்மையுடன் இருப்பேன் என்று உறுதியளிக்கிறேன். என் உடல் நலம் பாதிக்கப்பட்டாலோ, அல்லது எதிர்பாராத, வழக்கத்திற்கு மாறான நோய்குறி தென்பட்டாலோ உடனே இதை மருத்துவ அணியிடம் தெரிவிப்பேன் என உறுதி அளிக்றேன்.	☐

பங்கேற்பவரின் கையொப்பம் / ..... இடம் .....
கட்டைவிரல் ரேகை
பங்கேற்பவரின் பெயர் மற்றும் விலாசம் .....
ஆய்வாளரின் கையொப்பம் /..... இடம் .....
ஆய்வாளரின் பெயர் .....
மையம் .....
கல்வியறிவு இல்லாதவற்கு (கைரேகை வைத்தவர்களுக்கு) இது அவசியம் தேவை
சாட்சியின் கையொப்பம் /..... இடம் .....
பெயர் மற்றும் விலாசம் ....

SL. NO.	NAME	AGE	SEX	IP NO.	LIMB INV	CAUSE	GRD	CUL ORG	SENSITIVE ANTIBIOTIC	DOPPLER STUDY	PLAIN XRAY	RX	OUT COME	WOUNT MGT
1	Cehlla Pandiyan	62	M	62403	LL	DM	III	NG	-	MF in P	N	WD	Un	-
2	Piramudayan	75	M	62414	UL	DM	III	K,Pr,S	P, C, I, G, Ami	N	N	WD	W	SG
3	Karthikeyan	75	M	62479	UL	DM	II	Ps	P, C, I Ami	N	N	C	Un	-
4	Ramasamy	72	M	62827	UL	DM	IV	E	P, G, C, I	N	N	WD, F	W	SG
5	Chellaiah	64	M	71442	UL	CF	II	NG	-	N	N	C	Un	-
6	Mohammed Kani	63	M	74563	LL	RF	III	Ps	P, C, Cip, I	N	N	WD	W	-
7	Aminal	69	F	440	LL	B	III	K,Pr,S	P, C, Ami, G, I	N	N	WD, F	W	-
8	Rahaman Beeri	40	F	77543	LL	T	III	E	P, G, I	N	N	WD, F	W	-
9	Seenirasan	75	M	420	LL	DM	III	K	P, Cip, I	MF in PT	N	WD	W	-
10	Natarajan	48	M	77534	LL	UN	III	S, St	P	N	DEST TOE	A	D	-
11	Chandra	47	F	76096	LL	CF	III	K,Pr,S	P, C, I, Cip, G	N	N	WD, F	W	SG
12	Maharajan	50	M	37124	LL	B	II	NG	-	N	N	C	Un	-
13	Velmayil	75	M	72184	LL	DM	III	Ps	P, C, I, G	N	DEST TOE	A	D	-
14	Chithambaram	70	M	4456	UL	T	II	-	-	N	N	C	Un	SG
15	Esakki	58	M	2850	LL	IN	III	K	G, C	N	N	WD, F	W	-
16	Singaravel	65	M	2778	LL	T	II	S,St	P, C, I	N	N	WD	Un	-
17	Kutty Raj	40	M	71184	LL	DM	III	Ps	P, I, Cip	N	N	WD, F	W	-
18	Esakkimuthu	50	M	69650	LL	DM	IV	S	P, C	MF in P	N	WD, F	W	-
19	Vethasingamani Daniel	50	M	71180	LL	DM	III	E	Ami, Cip, C, I	N	DEST TOE	A	D	-
20	Subramaniyan	85	M	71259	UL	DM	II	S	P, C	N	N	C	Un	-
21	Govindan	50	M	69671	LL	DM	II	NG-	-	N	N	C	Un	-
22	Rajan	22	M	74494	LL	LY	III	S, St	P, C, I	N	N	WD, F	W	-
23	Ganesan	66	M	74525	LL	RF	III	NG	-	N	DEST TOE	A	D	-
24	Govindaraj	47	M	74515	LL	IV	III	K	P, G, I	MF in PT	N	WD,F	W	-
25	Subbiah	70	M	71252	UL	DM	II	NG	-	N	N	C	Un	-
26	Ramachandran	60	M	75862	LL	DM	III	K,Pr,S	P, C, I, G, Ami	N	N	WD	W	-
27	Arunachalam	62	M	75090	LL	DM	III	S,St	P, C, I	N	N	WD, F	Un	-
28	Kali	78	M	269	LL	DM	III	S	P, I, Cip	N	N	WD, F	W	-
29	Bakeer Mohamed	54	M	277	LL	DM	III	K,Pr,S	P, C, I, G, Ami	N	N	WD, F	W	-
30	Ramanathan	54	M	4887	LL	DM	III	S	P, C, I	N	N	WD, F	W	-
31	Seeniyesuradiyan	45	M	8205	LL	DM	II	NG	-	N	N	WD	Un	-
32	Murugan	64	M	17718	LL	DM	II	NG	-	N	N	C	Un	-

SL. NO.	NAME	AGE	SEX	IP NO.	LIMB INV	CAUSE	GRD	CUL ORG	SENSITIVE ANTIBIOTIC	DOPPLER STUDY	PLAIN XRAY	RX	OUT COME	WOUNT MGT
33	Lambert	47	M	19133	LL	DM	III	S	P, C, I	N	N	WD, F	W	-
34	Irulapasamy	42	M	23859	UL	DM	III	E	Amp, I	N	N	WD, F	Un	-
35	Ramadurai	58	M	23851	UL	DM	II	S	P, I	N	N	C	Un	-
36	]Sokkalingam	46	M	31824	LL	Un	III	NG	-	N	N	WD, F	W	SG
37	Patchathi	40	M	33747	LL	DM	III	E	P, C, Ami, Amp, I	MF in PT	DEST TOE	A	D	-
38	Arunachalam	62	M	33634	LL	DM	III	K,Pr,S,St	P, C, I, Cip, Ami	N	N	WD	W	-
39	Kumaradas	67	M	40282	UL	DM	III	E	P,C,I	N	N	WD	Un	-
40	Chelladurai	62	M	43442	LL	DM	III	Ps	P, C, I, Cip	MF in P	N	WD, F	Un	-
41	Kalimuthu	50	M	43444	LL	DM	III	K	P, G, C, I	N	N	WD	W	SG
42	Vellapandi	48	M	45127	LL	DM	II	NG	-	N	N	WD	Un	-
43	Patchaikani	75	M	57129	UL	DM	III	K,Pr,S,St	P, C, I, Cip	N	N	WD, F	W	-
44	Ibrahim	55	M	58130	LL	DM	III	S	P	N	N	WD, F	W	SG
45	Alphonse	56	M	58790	LL	DM	II	NG	-	N	N	C	Un	-
46	Lingam	65	M	58788	LL	DM	III	S,St	P, C, I	N	N	WD	W	-
47	Muthiah	58	M	58831	LL	DM	III	NG	-	N	N	WD, F	Un	-
48	Murugan	62	M	58873	LL	DM	III	S	P, C, Ciox, I	N	N	WD	W	-
49	Karuppasamu	40	M	58885	UL	DM	III	Ps	P, Ami, C, Cip, I	N	N	WD,F	W	-
50	Iyappan	50	M	60819	LL	DM	III	S	P	N	N	WD, F	W	-
51	Mydeen Pitchai	74	M	64041	LL	DM	II	NG	-	N	N	C	Un	-
52	Arumugam	54	F	63690	LL	DM	IV	K, Pr, S, St	P, C, Ami, Cip, I	N	N	WD, F	W	-
53	Fathima	47	F	63971	LL	DM	III	E	P, Amp, I	N	N	WD	W	-
54	Arumugaradivu	48	F	63999	UL	DM	III	S	P,C,I	N	N	WD, F	W	-
55	Muthiah	58	M	66347	UL	DM	III	NG	-	N	N	WD, F	W	-
56	Eswari	50	F	68914	LL	T	II	S	P	N	N	WD	Un	-
57	Arumugathamal	75	F	70181	LL	LY	III	K, Pr, S, St	P, C, Ami, Cip, I	N	N	WD, F	W	-
58	Ganapathi	55	M	71993	LL	B	III	NG	-	N	N	WD, F	W	-
59	Mariselvi	20	F	71997	LL	V	II	S	P	VI in SFJ	N	WD	W	-
60	Sornam	60	F	77043	UL	UN	III	ST	P	N	N	WD, F	W	-
61	Balasubramanian	60	M	78749	LL	RF	II	NG	-	N	N	C	Un	-
62	Rajakumar	32	M	78739	LL	V	III	K, Pr, S, St	P, C, Cip, I	VI in SFJ	N	WD	W	-
63	Pitchaiah	63	M	80221	LL	B	III	S	P, I	N	DEST TOE	A	D	-
64	Singarvel	65	M	2778	LL	T	III	NG	-	N	DEST TOE	A	D	SG

SL. NO.	NAME	AGE	SEX	IP NO.	LIMB INV	CAUSE	GRD	CUL ORG	SENSITIVE ANTIBIOTIC	DOPPLER STUDY	PLAIN XRAY	RX	OUT COME	WOUNT MGT
65	Chidambaram	70	F	4456	UL	DM	III	K, Pr, S, St	P, C, Ami, Cip, I	N	N	WD	W	-
66	Lakshmanan	75	M	7599	LL	RF	II	S	Ciox, I	N	DEST TOE	A	D	-
67	Balakrishnan	58	M	7716	UL	Un	III	NG	-	N	N	WD, F	W	-
68	Sunramaniyan	62	F	9274	LL	B	IV	S	P, I	N	N	WD, F	Un	SG
69	Indra	46	M	11022	LL	RF	III	NG	-	N	N	WD	W	-
70	Samsudeen	67	F	11907	LL	DM	II	K, Pr, S, St	P, C, Ami, Cip, I	MF IN PT	N	WD, F	W	SG
71	Ramalakshmi	60	F	12755	UL	CF	III	NG	-	N	N	WD	W	-
72	Mookandi	62	M	53878	LL	B	III	S	P, I	N	N	WD, F	Un	SG
73	Shanmugathai	54	M	14173	LL	DM	IV	K, Pr	P, C, Amp, I	MF in PT	N	WD, F	W	-
74	Veerakumari	55	M	14172	UL	T	III	S	P	N	N	WD, F	W	-
75	Velladurai	55	M	14256	LL	V	III	NG	-	VI in SFJ	N	C	Un	-
76	Velusamy	70	M	17604	LL.	T	II	S	P, I	N	N	WD, F	W	-
77	Shunmugasundram	65	M	17592	LL	B	III	K, Pr, St	P, C, Ami, Cip, I	N	DEST TOE	A	D	-
78	Chellappa	65	M	19384	LL.	T	III	NG	-		DEST TOE	WD, F	W	SG
79	Pommiammal	56	F	17632	LL	DM	IV	NG	-	NF in CALF VESSELS	N	BK	D	-
80	Murugan	50	M	52	UL	T	III	St	P, Ciox, I	N	N	WD	W	-
81	Chellappa	64	M	6304	LL	RF	III	St	P	N	N	WD, F	W	SG
82	Esakkiderar	80	M	6370	LL	B	II	NG	-	N	N	C	Un	-
83	Velsamy	60	M	9674	LL	CF	III	St	P	N	N	WD, F	W	SG
84	Madasamy	85	M	11221	LL	RF	IV	NG	-	N	N	WD	W	-
85	Mani	82	M	14429	LL	V	III	K, Pr, St	Ami, C, Cip, Amp, I	VI in SFJ	N	WD, F	W	-
86	Santhanam	67	M	23777	UL	IN	III	St	P, I	N	N	WD, F	W	-
87	Chelladurai	53	M	28395	LL	RF	II	NG	-	N	N	C	Un	-
88	Mohammed Hanifa	90	M	28525	LL	T	III	St	P, I	N	N	WD, F	W	-
89	Thambidurai	52	M	30074	LL	T	III	St	P, I	N	N	WD, F	W	-
90	Paramasivan	60	M	31634	LL	IN	III	S	P, I	N	N	WD, F	W	-
91	Chelladurai	60	M	34657	LL	T	II	St	P, C, Ciox, I	N	N	WD	W	SG
92	Murugan	92	M	38352	UL	B	III	NG	-	N	N	WD, F	Un	-
93	Kannan	35	M	40112	LL	T	III	St	P, I	N	N	WD, F	W	-

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94	Thangapandi	66	M	44881	LL	DM	II	K, Pr, St	P, C, Cip, Ami	MF in PT	N	WD	Un	-
95	Gurusamy	61	M	44910	UL	IN	III	S	P, C, Ami, Ciox, I	N	N	WD, F	W	SG
96	Sivasankaran	60	M	50517	LL	RF	IV	NG	-	N	N	WD, F	Un	-
97	Krishnan	65	M	50534	LL	B	III	K, Pr, St	P, I, Cip, Amp, Ami	N	N	WD	W	SG
98	Eswaran	60	M	50593	LL	T	III	NG	-	N	DEST TOE	A	D	-
99	Murugan	46	M	57069	UL	RF	II	NG	-	N	N	C	Un	-
100	Periyasamy	58	M	60532	LL	T	III	St	P, C, Ciox	N	DEST TOE	A	D	-